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OPTIC NEURITIS IN INTRACRANIAL DISEASE

Paper read before the Ophthalmological Society, March 10th, 1881

BY

J. HUGHLINGS-JACKSON, M.D., F.R.C.P., F.R.S.

Physician to the London Hospital and to the National Hospital for the Epileptic and Paralysed

(*Reprinted from* "MEDICAL TIMES AND GAZETTE" *March* 19, 1881)

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ON OPTIC NEURITIS IN INTRACRANIAL DISEASE.

Being a physician, my experience in any department of ophthalmology is necessarily one-sided. Optic neuritis interests me much as an important incident in many cases of intracranial disease, and comparatively little as an eye-affection. The wide bearings this condition has to physicians will be realised by anyone who will carefully study Gowers' valuable monograph, "On Medical Ophthalmoscopy." I divide what I have to say into several Sections—arbitrarily, I admit. It is not possible to write methodically on any subject so complex as this in mere linear order. The fact that optic neuritis may exist when sight is good will have to be considered in each Section.

Section I.—Optic Neuritis, Ophthalmoscopically.

I need not describe the appearances of the active process called optic neuritis. Although a continuous process, it varies exceedingly in degree at different times. I used, quite arbitrarily, of course, to make two stages—(1) slight œdematous swelling, almost limited to the disc; (2) a climax of extensive great swelling, with hæmorrhages and strangulation of vessels,—from either of which there may be retrocession to health; and two more stages in the going down to atrophy, the atrophy being properly a permanent post-neuritic condition. For my own part I think there is but one kind of optic neuritis from intracranial disease. It seems to me that the young ophthalmoscopist supposes there to be varieties, because the appearances at different times differ so vastly. And some of those distinguished men who make the distinction into "choked disc" and "descending neuritis" admit that ophthalmoscopically it is often difficult to tell

one from the other—that there may be mixed conditions. To avoid begging the question, let me use, when the possible distinction is important, the general term Swelling of the disc. It is certain that swelling of the disc occurs in many different affections; and in this way we might speak of varieties. To mention some. It is found in some cases of Bright's disease, usually the granular kidney; it is found in most cases of intracranial tumour; it is found in some cases of meningitis; and it is particularly to be mentioned that it is found in some cases, clearly from the symptoms cases of intracranial disease, where ordinary examination (post-mortem) discloses no local disease within the cranium or in any other part of the body. Hulke, Soelberg Wells, and other ophthalmic surgeons speak of a recoverable optic neuritis in young women suffering from uterine derangement. There is, as Hutchinson has shown, swelling of the disc in some cases of lead-poisoning—in one case appearances quite like optic neuritis from intracranial tumour. (The presence or absence of renal disease should be noted in all records of these cases.) I have seen optic neuritis like that from intracranial tumour in one case of diabetes, but there was found post-mortem local organic brain disease, remains of a clot. It is only possible to consider some points which should come under notice in this Section.

Bright's Disease.—In most cases there is no difficulty in declaring correctly that certain ophthalmoscopical appearances signify Bright's disease, even if we ignore the ordinary evidence of renal disease. At any rate, as a matter of fact, an ophthalmic surgeon often correctly predicts Bright's disease after an ophthalmoscopical examination only. But in a few cases the appearances of the fundus are exactly those in some cases of intracranial tumour without Bright's disease. For in each there may or may not be the well-known white streakings in the region of the macula; in each there may be great swelling of the discs, and blotches, not only streaks, of blood. Moreover, the general symptoms—I mean intense headache and vomiting—may be alike. The general course of the case is often the same. The patient may be a child. There is nothing by which we can distinguish, except, on the one hand, by an examination of the urine and vascular system, or unless, on the other hand, there be certain localising symptoms. Such symptoms may be, and very often are, absent early in the cases. The localising symptom, hemiplegia, with or without apoplexy, might, however, be either an indirect result of the constitutional state in Bright's disease, or might signify hæmorrhage

from a cerebral tumour; the most trustworthy localising symptoms helping the diagnosis of tumour are such as unilaterally beginning convulsion and paralyses of cranial nerves.

But even yet there are difficulties. I have seen double optic neuritis in a man who had unilaterally and deliberately beginning convulsive seizures—a condition pointing mostly in such association to cortical tumour—and Bright's disease too. I saw a case many years ago, under the care of Dr. Habershon, in which there were found post-mortem both renal disease and intracranial tumour, although in this case intracranial disease was diagnosed by other nervous symptoms.

Brudenell Carter wrote some years ago :—"We had a little boy in St. George's Hospital in 1872, who was transferred from my care to that of Dr. Fuller, and who had choked discs of the most typical character. Dr. Hughlings-Jackson saw him, and entertained no doubt that he was the subject of some form of brain disease." This is quite correct. I suppose I stupidly omitted to examine the patient's urine. Mr. Carter continues—"and the same opinion was expressed by Dr. Noyes, of New York, and by several members of the International Ophthalmological Congress which was then assembled in London. The boy died of pleurisy supervening upon advanced kidney disease, and no trace of mischief in his brain could be discovered by the most careful examination." Then, illustrating the difficulty otherwise, Mr. Carter continues—"Nearly at the same time we had in the hospital a young woman whose eyes presented typical examples of the changes often associated with albuminuria, but who died with healthy kidneys, of a tumour in the cerebellum."

Are there any recent researches which teach us to make a diagnosis by the ophthalmoscope in such a case as the following ?

Suppose a patient with Bright's disease has hemiplegia, and suppose his fundus presents none of the well-known changes once supposed to be characteristic of Bright's disease, but appearances which are very common in cases of cerebral tumour, can we tell whether the neuritis is an outcome of Bright's disease or is secondary to the clot which the hemiplegia implies? I used (1865) to put down these cases to the latter cause; now I do not conclude on the matter. I now doubt whether I have ever seen double optic neuritis from clot, although I have seen the two things coincident.

What is the pathogenesis of the changes when no local organic disease is found in the head post-mortem ? It has been suggested that the common changes in the fundus

in cases of Bright's disease are directly consequent on changes in the brain occurring with kidney disease.

Meningitis.—I speak of three kinds of meningitis: 1. *Tubercular meningitis.* In doubtful cases, swelling of the discs is, at any rate, of great value in helping to the diagnosis of some kind of acute intracranial disease. But, as a rule, it comes on in tubercular meningitis at a time when diagnosis is made from other evidence. In tubercular meningitis the swelling is slight, not extensive; it is even, merges into the fundus; the disc is succulent-looking; there is not time, possibly, for further development. It is like the earliest stage of optic neuritis from intracranial tumour. 2. *Traumatic meningitis.* Of swelling of the discs in non-tubercular meningitis of any kind I know scarcely anything. I have seen meningitis without any such swelling, even universal meningitis like that following injury to the head. *Cerebro-spinal Meningitis.*—I do not remember seeing swelling of the discs in any case of this kind, but it sometimes occurs. Dr. Stephen Mackenzie reminds me of two cases.

At this juncture a clinical difficulty must be mentioned. Intracranial tumour sometimes produces an acute illness, which may be fatal, not distinguishable by its symptoms from meningitis, tubercular or traumatic.

So it might be a matter of importance in diagnosis to know what particular ophthalmoscopical signs there are to be trusted in the differential diagnosis, apart, of course, with regard to tubercular meningitis, from seeing tubercle in the choroid. I know of none myself beyond degree and inferred long duration of changes.

What signs, if any, enable us to distinguish betwixt swelling of the discs in the three kinds of meningitis?

What is the frequency of occurrence of changes in the discs, and their kind, in traumatic meningitis and in the allied meningitis from ear disease? In some of these cases there is pyæmia too. When swelling of the disc is found in these cases, is it part of, or a result of, the meningitis, or is it ever a result of pyæmic blocking of veins? Is there thrombosis of the sinuses in any of these cases?

Can we tell the swollen disc in cases of meningitis from optic neuritis (in its early stage) owing to intracranial tumour?

Mentioning, again, that a cerebral tumour may produce an acute illness like meningitis, I would suggest that evidence from cases uncompleted by autopsy, or unless the patient recovers, is not of great value towards answering the questions just put.

It is well known that sight may be good in severe neuritis, and that the neuritis—under treatment, perhaps without—may pass off. Although it is common and convenient sometimes to speak of optic neuritis as a symptom, it is really a pathological condition. We have just said, in effect, that there may be a pathological condition of the discs without the symptom, amaurosis. Do these cases differ essentially from cases in which sight is defective or lost? I think not. Some say that the former are cases of "choked disc," and the latter of optic neuritis. Is this an inference from the condition of vision, or is the distinction to be made by the ophthalmoscopical appearances? I know of no difference in *kind* of disc betwixt cases in which sight is good and those in which it is defective or lost, but of difference in *stage* of changes only.

It is known that optic neuritis may pass off under treatment, leaving sight good, whether there has been any defect of sight or not, even when there has been complete blindness. It may be an important question as to diagnosis of any present ailment to ascertain that there *has been* neuritis. I see discs in patients whom I know to have had acute neuritis, which, were I seeing the patients for the first time, I should not dare to declare abnormal. But, supposing that optic neuritis always does leave some traces discoverable by direct examination, I put the *question:* What are the ophthalmoscopical signs by which we may ascertain in such cases as those alluded to that there *has been* optic neuritis? I do not mean only when we are told that the patient has had defect of sight, for there may have been neuritis without any defect of sight. I have seen many cases, and have recorded several, in which the relics of past well-marked neuritis were trifling. As the expression "trifling" is vague, I will mention a case, choosing, for obvious reasons, one in which the examinations were made by an ophthalmic surgeon. On June 12, 1879, Mr. Couper saw a patient of mine who had normal acuteness of vision, normal accommodation, and normal field. "At the same time, there is well-marked double optic neuritis." "The state of the discs accurately corresponds to that often associated with brain mischief." About nine months later, Mr. Couper reports:—"March 24, 1880: I re-examined yesterday, and found the translucency of both discs perfect. Their colour, although somewhat full, is within normal bounds. By direct examination, the redness of the left disc is seen to be somewhat patchy, and there is a little more projection of nerve-substance than normal. Both these conditions are absent in the right disc. There

are no thickened sheaths, except a trace in the case of one retinal vein on the left disc. The maculæ and their regions are healthy and *free from greyness and œdema*. Either his accommodation was unduly active, or a slight myopia has developed in the right. It requires −72 spherical to get $\frac{20}{50}$. Even the left got $\frac{20}{30}$ more easily with −72. The amount of myopia, even if real, is of no importance. It thus appears that a very satisfactory improvement has come in the discs and retinæ."

Not unfrequently patients come to physicians for such a nervous symptom as localised convulsion, giving a more or less vague account of some illness with headache and vomiting months before. The discovery of relics of past neuritis might help us in determining whether the symptom we are consulted for depends on gross organic brain disease or not.

Let me put the matter in another way. It is stated by Mr. Couper that prolonged use of the eyes in some cases of hypermetropia induces œdema and swelling of the discs, occasionally mistaken for, and treated as, slight optic neuritis depending on cerebral disease. Is it possible that swelling of the discs thus caused may leave behind it traces like those left on what we call recovery from neuritis?

Unfortunately, the discs do not always go back to health, but, so to speak, down to atrophy, partial or total. In most cases we can tell by the ophthalmoscope whether the atrophy is the sequel of some neuritis, although, maybe, not that there had been the neuritis which is commonly called optic neuritis. And, if we could not diagnose past neurosis, we could in most cases infer it. But I am not speaking of inference. The question I put is this :—

What are the best ophthalmoscopical signs to rely on in the diagnosis betwixt atrophy after a neuritis and the atrophy in locomotor ataxy, disseminated sclerosis, etc., apart from collateral evidence? The signs generally given as diagnostic of past neuritis are veiling of the lamina cribrosa, white marks along the vessels, ill-margined disc, irregularity of veins. Are such signs always present in very long standing cases of post-neuritic atrophy?

[I omitted speaking of conditions of the pupils in cases of double optic neuritis, and now I speak on but one point. Supposing, when blindness, with atrophy of the discs, has come on, the pupils do not act to light, do they act during accommodation? I have seen two patients, each with mere perception of light, in the different atrophy of tabes dorsalis, whose pupils did not contract to light (which is not perhaps remarkable), but did contract well during accommodation.

The patient is told to look at the end of his nose, or at his hand held near his face. The "Argyll-Robertson condition" was as manifest in these two amaurotic patients as in any case of tabes dorsalis, without optic atrophy, I have seen.]

Section II.—Clinical Facts.

Optic neuritis is usually double—in physicians' practice, nearly always. I have seen but two cases of intracranial disease (proved post-mortem, I mean) in patients who had uniocular neuritis. In each case there was a tumour of the opposite cerebral hemisphere, and in each hemiplegia of the same side as the neuritis. Both eyes from one of the patients were examined microscopically by Dr. Hermann Pagenstecher, of Wiesbaden. The left eye, on the side of the tumour, was found by him to be normal, as it had been declared to be during the patient's life.* These cases are very exceptional. It is notorious that tumour of but one cerebral hemisphere, nearly always, if it produces optic neuritis, produces *double* optic neuritis, and, what is more to the point, the neuritis begins nearly at the same time on the two sides; it may be unequal in degree; there is often great difference in the sight, at least.

What does uniocular optic neuritis signify? Has it been found in cases of tumour of one cerebral hemisphere which had not produced any localising symptoms? I put this question for clinical reasons, and with regard to the next—Does uniocular neuritis result from pressure or involvement of the optic nerve? Stating the last two questions clinically—If a patient has uniocular optic neuritis with severe headache and no ordinary localising symptoms, how are we to tell whether he has disease at the base or in one cerebral hemisphere? Does uniocular optic neuritis ever result from local organic disease in the cerebellum? I have not seen this, although I have often seen it double from disease of different parts of the cerebellum. Can we distinguish uniocular neuritis resulting from tumour or aneurism in the orbit or near the cavernous sinus inside the cranium, from uniocular neuritis from disease of the great centres—of course, I mean apart from there being palsies of parts supplied by nerves entering the orbit? Is there anything in the degree of swelling to be relied on?

I feel convinced that the diagnostic value of optic neuritis is not different whether sight be good or lost. My own

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* *Royal Lond. Ophth. Hosp. Rep.*, November, 1871.

impression is that there is nearly always a stage of neuritis before sight fails. I see more cases of it with good vision than with even defect of sight. Gowers told me some years ago that in at least half the cases he had seen there had been no defect of sight. Dr. Stephen Mackenzie's experience agrees with mine. When no distinction is made betwixt the pathological condition and the symptomatic condition, the statement that optic neuritis may exist when sight is good seems to be taken as equivalent to saying that with defect of sight there may be no defect of sight. The following quotation bears interestingly on the matter, showing how different fields of work give seemingly different results :— "The cases of intracranial disease which come before me as an ophthalmic surgeon have all more or less impairment of vision—on account of which I am consulted. On examination, I find either optic neuritis or its sequel, atrophy of the disc. But if I examine cases in the medical wards, I often find ischæmia of both discs in cases where there is no want of sight" (Higgens, *Guy's Hospital Reports*, 1875). Mr. Higgens has evidently considered the question carefully and broadly. I do not agree with him in his conclusion that there are two different conditions of the disc.

When sight fails, at what stage does it fail? Ten years ago I—perhaps rashly—said that a late stage of incipient clearing-up was the one most often seen by those who do not look at the fundus until sight begins to fail. I think an ophthalmic surgeon and a physician would give very different accounts of the earliest appearances of optic neuritis, if each kept rigidly to cases in his own department. Physicians often see it in the pre-amaurotic stage.

Drawings of optic neuritis often represent a late stage of optic neuritis, being made from discs when sight has failed or is failing. Any drawing of optic neuritis is only really of some stage of the process. Yet sometimes there is no failure of sight when neuritis has persisted some months, and when the discs show the late changes sometimes called "woolly disc."

Does sight sometimes fail before any neuritis is discoverable—before it appears intra-ocularly? Are these cases of pressure on the optic nerves? Some of these patients might be supposed to be hysterical. A fine-looking young woman came to Moorfields many years ago, *complaining of her sight*, but could read "brilliant" type. Among others, Bader examined the discs and found no abnormality. Some thought her hysterical, although she said she had had intense headache for three weeks. A week later neuritis came on (or rather, I suppose, appeared intra-ocularly). I

saw her in Guy's a year later, blind from optic atrophy, and hemiplegic of the left side. She died later on in another hospital; all I could get to know of the necropsy made was that "induration of each cerebral hemisphere and of the spinal cord" was discovered. Mr. Hutchinson has mentioned to me cases of similar bearing.

Temporary Total Failure of Sight.—In some cases of optic neuritis the sight fails for a time totally or partially, although at most times it is good. The patient may be able to read the smallest type, and yet occasionally, for a few minutes, becomes quite blind.

What do these sudden and temporary failures of sight in neuritis, with good vision, signify? Do they occur most often when the disease is of the cerebral hemisphere? Do sudden temporary total failures of sight, or—the functionally opposite thing—projections of colour, sometimes precede neuritis? Excluding cases of migraine, I should fear they heralded neuritis if there were intense pain in the head. (I used to speak of these cases as "epilepsy of the retina," but have for many years abandoned that term.)

Rapid Permanent Failure of Sight.—Sight being for some time good in neuritis, may fail very rapidly—in a few hours. Such failure has, in my experience, been ascribed by patients to instillation of atropine, to galvanism, to blisters. Hence the importance of recognising such occurrence.

Do the ophthalmoscopic appearances change much at the time when rapid failure occurs? Of course, there must be some change, but I have not been able to appreciate it. What is the significance of the rapid failure?

Section III.—Association of Optic Neuritis with other Symptoms.

The first remark is, that good general health goes practically for little in our estimation of the gravity of the situation in many cases of optic neuritis, even when sight is good. We all see healthy-looking, robust children and adults, who have double optic neuritis with good sight, who are sometimes supposed, by those who do not use the ophthalmoscope, to have no intracranial disease at all. Some of the patients, nevertheless, have intracranial tumour; sometimes death occurs rapidly and unexpectedly. It is safest, although illogical, to take good general health with optic neuritis as going for nothing for prognosis in young people. They may have vascular gliomata, and if so are always on the brink of death.

Now, as to symptoms in the usual sense of the word. These may almost be said to divide themselves into two groups—localising and non-localising.

Optic Neuritis, with Non-Localising Symptoms.—In most cases optic neuritis goes with headache, and often with vomiting; all three belong to one clinical group. Very often these symptoms come first, or are the sole symptoms up to the end of the case. Sometimes the headache precedes the neuritis, and—what is quite a different statement—very often indeed it precedes failure of vision. The three symptoms depend in very many cases on local gross organic disease within the cranium, but are no further localising; they do not help us to determine in what particular part within the cranium the inferred disease is. A more strictly correct description of the three symptoms would be to call them signs—the best signs, at least—of local gross organic disease within the Cranium; but in this connexion, non-localising is the more convenient name.

The headache is intense; often it is paroxysmal, the patient being in the intervals apparently quite well. The vomiting is purposeless; and bile is often brought up. Hence the frequent, almost invariable, diagnosis in the early stages of chronic cases of cerebral tumour (when sight is good, or only slightly defective) of liver derangement, and in acute cases the occasional diagnosis of "bilious or gastric fever." I suppose ophthalmic surgeons often see children blind with optic atrophy, said to have lost their sight after "bilious disorders." Some years ago, Hutchinson wrote on this matter. The symptoms headache and vomiting very often indeed occur when there is neuritis without defect of sight; the patient in the intervals of his pain may go about, and even, if the pain comes on only at night, transact his business; he reads the newspaper as usual. The patient is supposed to be only bilious, or sometimes, if a woman, to be only hysterical, until some unmistakable intracranial symptom, such as convulsion, suddenly alters the diagnosis. Not unfrequently the patient, supposed to have a slight ailment, dies rapidly, or even suddenly. Some patients who have become blind, or otherwise incapacitated, complain bitterly of the errors in diagnosis made early in their cases. The routine use of the ophthalmoscope would save us from many of these errors in diagnosis. No one would make the diagnosis of liver disease nor of hysteria *only*, if, looking at the discs, he saw optic neuritis.

As already said, there is sometimes an acute illness as our first evidence of tumour. But there are degrees up to this

from great chronicity, with slight accentuation of symptoms. It is very important to bear in mind that optic neuritis may occur without any headache and without vomiting, or that the headache may be slight and the vomiting none. Another way of putting what has been said is to say that there are all grades of severity of the ailment, of which optic neuritis forms one part, from a condition which (when sight is good, as it often is) is mistaken for digestive derangement, hypochondriasis, or hysteria, up to a condition symptomatically like that of tubercular meningitis.

At this juncture we may most conveniently consider cases after neuritis has passed off, leaving or not leaving optic atrophy. First let us instance a chronic case. A patient comes to us for severe headache and vomiting—not, I am supposing, for defect of sight, there being none. We discover double optic neuritis, and, from a survey of the whole case (in spite of the good general vigour of the patient, and regardless of the fact that at times he feels well, and may then attend to his business), we declare him to have some kind of serious intracranial mischief. Yet, after taking iodide of potassium, he gets apparently well, sight at no time being affected. One opinion on the case after the recovery is that the diagnosis made was wrong—disproved by the recovery. For the doctrine that a patient who says his sight is good, and whose statement is verified by testing it, has double optic neuritis, is not seldom considered to be simply "moonshine." To some a more reasonable explanation seems to be "liver," etc.

Suppose, next, that there was an acute illness, from which the patient has recovered, except for defective sight, with some atrophy of his optic discs. Here comes the question, Was the illness in either of the cases meningitis? For my own part, I believe it to be more probable that it was, and remains, a case of local gross organic disease, such as tumour. It is of no avail to say of the acute case that the patient had "all the symptoms of a meningitis," for there are no symptoms known to be characteristic of meningitis only. Admitting that, during the illness, we might be unable to decide, I should take the mere fact of recovery as strong evidence against meningitis—not that I need deny that recovery from meningitis occasionally happens. Virchow speaks of recovery from tubercular meningitis. Autopsies alone can decide. So far as autopsies go (had some time after recovery on return of head symptoms, or after death by some other cause), the evidence is in favour of local gross organic disease rather than of meningitis, in the kind of cases referred to. I feel

sure, at any rate, that facts show that even complete recovery with good sight does not negative persisting local gross organic disease within the cranium. This is strikingly true of some cases of syphilitic tumour; the symptoms pass off under treatment, but the patient may come again and again for the same localising symptom, showing disease persisting in one place, as in a striking case (of which I am thinking) the necropsy proved. So, instead of asking for evidence from cases of patients who have recovered from symptoms of meningitis, I ask the question, Have traces of meningitis been found after such recoveries, when patients have died later on of some other disease?

Prompt recovery from such conditions under anti-syphilitic treatment is not, as a matter of fact, to be confidently explained on the supposition that the case was one of syphilitic brain disease.

From any one of or from all of the three symptoms occurring either acutely or chronically we cannot be certain of tumour, but in a chronic case we are in most cases right in predicting it.* Suppose we were to feel certain of the existence of tumour, we should have, from the symptoms mentioned, so far as I know, not a particle of evidence as to the locality of the tumour beyond that it was intracranial; hence I have called the symptoms non-localising. As this is simply from my ignorance, I put the question, Are there any means of ascertaining the position of inferred local intracranial disease by any peculiarities in the neuritis, headache, and vomiting? (Of course, if the headache be persistently one-sided, it is then a localising symptom.)

Does the degree of pain and vomiting depend especially on the acuteness of the case or on physiological localisation —that is, on great involvement of grey matter,—or on both?

Here is a convenient place to speak of rapid or sudden death in cases of optic neuritis (without Bright's disease), without other marked symptoms than headache and vomiting. In some cases the patient dies rapidly; this may be by hæmorrhage from a tumour, when apoplexy and hemiplegia, or sudden convulsion, may occur. He may die suddenly, when in intense pain; this has happened in a case of cerebellar abscess from ear disease, which had not ruptured. Intense persisting pain in cases of optic neuritis makes me fear that the patient may die suddenly. But sudden death occurs in cases of intracranial tumour without much pain, and without hæmorrhage from it. We should

(*) It is understood, of course, that I do not assert that uniocular neuritis is non-localising.

never lose sight of the fact, that a patient with optic neuritis and other non-localising symptoms may die rapidly or suddenly of the brain disease which they signify. But, besides rapid or sudden death in patients acutely ill (which may not seem remarkable, although in the cases I allude to it was to me most unexpected), patients with optic neuritis who have, to superficial appearance, little the matter with them—those supposed to be bilious or hysterical—will die rapidly or suddenly. The most striking cases are those of patients who have no defect of sight with their optic neuritis; and in some of them I feel convinced the diagnosis of intracranial disease could not be reasonably made without using the opthalmoscope. A man whom I thought to be hypochondrical until I looked into his eyes, who gave only a vague history of twitching of one lip, had double optic neuritis, with good vision. He seemed, superficially regarded, to have nothing the matter with him; he became suddenly apoplectic, with hemiplegia, and died. A woman who had double optic neuritis, with good vision and some other vague nervous symptom, got well, except that the neuritis remained. Vision being still good, she then had attacks of giddiness, in some of which she would fall. She, however, looked perfectly well, and yet died in a few minutes one night; the evening before she was out with her husband marketing. There was an inquest, but no necropsy. A patient with double optic neuritis is always in imminent danger, however well he may seem in general health. Again, after seeming complete recovery from severe intracranial symptoms, optic neuritis remaining, I have known death occur rapidly by convulsion from hæmorrhage from a vascular tumour. I have known a girl who got well except for amaurosis from optic atrophy after neuritis, who died in the night by hæmorrhage from a cerebral tumour.

Optic Neuritis * *with Localising Symptoms.*—Localising symptoms are of no rational value, perhaps of some empirical value, towards the diagnosis of gross organic disease. They point to position alone. It is impossible to go into this part of my subject except briefly. It is exceedingly complex. We are now supposing that, from what we have called the non-localising symptoms, we infer tumour.—I would first remark, however, that there may be no localising symptoms, indeed, no striking symptoms of any sort when there is a tumour, even a large one, in the cerebrum or cerebellum.

* Only a fragment of what was written under this heading was read.

(*a.*) *Unilaterally* beginning convulsion—in the hand, in the side of face or tongue, or both, or in the foot—followed or not followed, by temporary hemiplegia, points to disease involving some part of the cortex cerebri, usually in the mid-region of the brain.

(*b.*) Slowly coming on hemiplegia of the ordinary form points to the cerebrum, and is strong additional evidence of tumour.

Question.—Are not optic neuritis and headache often absent in this variety of hemiplegia, or late in coming on?

(*c.*) If, in a child with slowly coming-on hemiplegia, there be great enlargement of the head, there is a voluminous adventitious product of one cerebral hemisphere.

(*d.*) Sudden hemiplegia may betoken hæmorrhage from a cerebral tumour, which hæmorrhage, if it be plentiful, may produce apoplexy too. Unless there be optic neuritis, or a clear history of intense headache, these cases cannot, in persons past middle age, be distinguished from cases of ordinary cerebral hæmorrhage. Even when there is optic neuritis, there will then be Bright's disease to be considered—the difficulty stated in Section I. In the two cases of uniocular neuritis already mentioned, the neuritis was the only thing I relied on for the diagnosis of tumour as a cause of the hemiplegia in each.

(*e.*) Reeling gait points to tumour of the cerebellum, or to tumour under the tentorium. If in children there be great increase of size of the head, the tumour is probably of the middle lobe. In these cases there may be tetanus-like attacks, and later there may be, also, continuous universal rigidity, mostly with retraction of the head.

(*f.*) *Affections of Cranial Nerves.*—In my practice, only that of a physician, I rarely see double optic neuritis complicated with paralyses of the cranial nerves. When cranial nerves are paralysed, we have to distinguish into (1) Cases where certain of them are paralysed along with hemiplegia, and then whether the grouping points to tumour of the pons Varolii, or of the crus cerebri, or to tumour pressing on the crus cerebelli. The symptoms may be so grouped as to point to one lesion, and if they come on slowly together the diagnosis of tumour is, without other evidence, almost certain. When they cannot be so grouped—as, for example, when paralysis of the parts supplied by one third nerve is on the same side as paralysis of the arm and leg—there are two lesions, and the diagnosis of intracranial syphilis is

favoured. (2) When paralysis of widely separated cranial nerves occurs there is usually intracranial syphilis, but occasionally cancer of the base. (3) When there is such a grouping as paralysis of the third, fourth, and sixth nerve with optic neuritis or atrophy, there is probably tumour or aneurism at the orbital fissure.

Non-associations.—Optic neuritis is scarcely ever, if ever, found with ordinary epilepsy—the epilepsy of nosologists. It is very common with epileptiform seizures—convulsions beginning deliberately in some part of one side of the body. There are, however, a few cases of gross organic cerebral disease in which there are paroxysms like those of epilepsy proper, and occurring as in cases of epilepsy proper at intervals, health being good betwixt. If double optic neuritis be found in a case which symptomatically is like epilepsy proper, I should surmise that there was gross local organic disease. I have never seen optic neuritis in chorea. It is particularly to be remarked that optic neuritis is rare with complete persistent aphasia; not very uncommon with slight aphasia, nor with temporary complete aphasia after right-side-beginning epileptiform seizures. The reason, I suppose, is that complete persistent aphasia often depends on clot or softening. On the other hand, tumour, the common cause of optic neuritis, is a very rare cause of complete aphasia. My impression is that most of the cases reported as exceptional to Broca's localization are cases of tumour in which I may call Broca's region. It is rare to see optic neuritis associated with extreme positive mental symptoms. In two cases I have seen the patients so affected become maniacal; in neither was there any local gross organic disease discovered post-mortem. It is very striking that whilst tumours of the cerebrum or cerebellum often produce defect or loss of sight of both eyes, although in an indirect way, they never, so far as my experience goes, produce deafness of either side in any way, with the exception of tumours pressing on the auditory nerve, and those producing greatly raised pressure under the tentorium. Loss of smell is more common, but I have nothing definite to say on frequency of association of anosmia with optic neuritis. I know of no evidence to support the hypothesis that optic neuritis is ever caused in any way by disease of the nervous system below the cranium.

Section IV.—On the Diagnostic and Non-diagnostic Value of Optic Neuritis.

Optic neuritis, although so often found with disease of the cerebrum or cerebellum, does not occur from mere destruction of any particular part of the encephalon. We are not here concerned with the causation of the *negative symptom*, defect, or loss of sight, but with the process of production of the *positive pathological condition* (optic neuritis) upon which condition defect or loss of sight may or may not ensue. *A priori* one could not expect that a merely destructive lesion—a negative condition—could cause optic neuritis—a positive condition; nothing cannot cause something. Hence to bring evidence in support of the dictum seems needless. But the term "disease of the brain" is often used loosely in these and in other cases of nervous disease. It is proper to show clearly that destructive action by a tumour is only one of its ways of acting. It is not at all uncommon to hear positive symptomatic conditions spoken of as being owing to negative states of parts of the nervous system; for example, lateral sclerosis is said to "cause" rigidity in hemiplegics. Again, negative lesions do, I think, often enough indirectly cause, or rather they permit, positive symptoms, as in the case just instanced. And after all, to adduce facts in favour of the seeming truism will enable us to group them methodically for a further purpose.

Optic neuritis mostly occurs with intracranial tumour or some other adventitious product. The mass may be in any part of the encephalon, anterior, middle, or posterior lobe of cerebrum, pons Varolii, cerebellum (middle lobe, or right or left lobe); it may be at the base. Like Gowers, I have not yet seen optic neuritis with tumour of the medulla oblongata; I should say that I have only once seen tumour limited to that part, and forget whether there was neuritis or not. It may properly be said that tumours destroy little; but they do destroy a little, and by induction of softening about them they often cause much destruction.

So I first assert that tumour in any part of the encephalon, with the possible exception of the medulla oblongata, may produce double optic neuritis. This is in entire harmony with the statement in Section III. that optic neuritis is of no localising value.

Next, optic neuritis may not be found with tumours or other masses in different parts of the cerebrum. It may not be found with adventitious products in the cerebellum. I have seen a tumour in each lobe of the cerebellum, and also one in the left cerebral hemisphere, in the case of a patient

who presented no trace of acute neuritis, but slight changes, doubtfully to me indicating long past neuritis. Hence my interest in the answer to a previous question on relics of past neuritis. I have known neuritis absent in a case of abscess of one lobe of the cerebellum.

So now, taking in the previous statement, I say that optic neuritis occurs with tumour in any part of the encephalon, and may not occur with tumour in many parts of it. A further fact in complete accordance is, that optic neuritis may occur late in cases of cerebral tumour. This is, of course, in one way an important qualification of a previous remark (see preceding paragraph), but does not invalidate—on the contrary, enforces—the general statement we set out with: that optic neuritis is not caused by mere destruction of any part of the encephalon. A man died under my care, in March, 1875, of tumour of the left cerebral hemisphere, who had had convulsive seizures beginning in his right foot nine years: presumption, considering the correspondency of this symptom with the part found diseased post-mortem, of the existence of tumour for nine years. Yet his discs were normal until a few weeks before death. So late as December, 1874, when Mr. Couper examined them, finding a high degree of hypermetropia, they were normal. He found neuritis about the middle of February, 1875.

Then optic neuritis may appear late and pass off, the patient dying ultimately of tumour. A man was under my care first in December, 1873. It was only on September 19, 1874, that changes in his optic discs were seen. His discs had been examined scores of times before, the diagnosis of tumour and of its general position having been made months previously. Now, I saw this man in August, a month before the onset of the neuritis, in a condition which I thought would probably soon end fatally; had it done so, the record would have been of a case of tumour of the brain which had not produced neuritis. As the actual progress of the case showed, the statement, "which had not *yet* produced neuritis," would have been better, for, as I have said, it did come on later. In about six weeks after the onset of the neuritis the discs were again normal. Had I examined for the first time the discs only shortly before death, which occurred in December, 1874, I might have supposed too that the tumour had not produced optic neuritis. This case, as do many others, teaches us to examine the eyes of our patients repeatedly.

Optic neuritis exceedingly rarely occurs in cases of extensive destruction of brain by softening or clot (hence it is rare with complete aphasia and with ordinary hemiplegia, or

both together). I have already spoken of clot in the brain in cases of Bright's disease, and of course I am just now leaving out of account cases of hæmorrhage from cerebral tumours. There are at least two cases of double optic neuritis in connexion with local softening, from embolism or thrombosis (Broadbent and Stephen Mackenzie). Softening and clot affect a particular locality; and it might be said that in this is one reason for there being with them so rarely any neuritis. But it is sometimes found with tumour in the region so frequently affected by clot and softening.

Additional evidence quite in harmony with the very different evidence already adduced is that optic neuritis may exist when sight is good. And still further evidence having the same tendency is that, as already illustrated, optic neuritis may pass off when the gross disease causing it remains.

To resume. Because optic neuritis occurs with tumour of any part, and may not occur with tumour of many parts, because it comes on late in some cases of tumour, and passes off again in some, and because it is very rarely found with such widely destructive processes as softening and clot, and because it may not be attended by any defect of sight, it is inferred that it does not depend on destruction of any part of the nervous centres.

Nevertheless, there is a relation of some kind betwixt the tumour or other gross product and the neuritis. The relation is indirect; the tumour produces neuritis secondarily. Before we can discuss the nature of this secondary process, I must speak more precisely than I have yet done in using for the most part the word "tumour."

Optic neuritis points to the general nature of the local disease, not to its particular nature. It is mostly produced in cases of gross local organic disease, but that may be not only tumour ordinarily so called, or syphiloma, but cyst or abscess—may be an adventitious product of almost any kind. We may safely make the generalisation that it points, most often, to a "foreign body" of some kind—of almost any kind, not to any one in particular. If a magician were to introduce a lump of wood into any part of a man's encephalon, it might, I believe, produce double optic neuritis like that produced by any "foreign body" of pathological origin. It may be well to remark expressly that a syphilitic "lump" in the brain produces just the same kind of neuritis as any other sort of lump in it does. "Optic neuritis induced by syphilitic disease" of the brain is not "syphilitic optic neuritis"; it differs in nothing essential from neuritis induced by a cortical glioma, which neuritis we should not

call "gliomatous optic neuritis.' Hence the vagueness of the superficially seeming definite expression "optic neuritis *caused by* syphilis." The defect of sight which goes with intracranial syphilis in cases physicians see, if associated with changes in the fundus, is scarcely ever dependent on choroido-retinitis; it nearly always occurs with optic neuritis. (I may here remark parenthetically that I have never seen a syphilitic neuroma of the optic nerve or tract.)

There is no difference in the neuritis produced by tumour of any kind, by abscess, by cyst, or by almost any other foreign body.

This statement does not imply that some adventitious products may not more often produce optic neuritis, or produce it sooner than others, or produce a greater degree of it more rapidly. It is believed by some that vascular gliomata produce more headache, and more paroxysmal headache, and altogether a more acute disturbance, than, for an example, an indolent mass of tubercle.

Optic neuritis, as several times remarked, very rarely, if ever, occurs from clot in the brain. Here it should be noted that it is possible to mistake hæmorrhage from a vascular cerebral tumour for ordinary cerebral hæmorrhage—it is possible to overlook the tumour; the hæmorrhage is conspicuous, the tumour may be inconspicuous. Virchow speaks of one case in which only a careful microscopical examination enabled him to discover that there was tumour in a case of cerebral hæmorrhage. Hence I need feel little ashamed in confessing that one case I have recorded of optic neuritis with other symptoms, as being owing to ordinary cerebral hæmorrhage, was, I doubt not now, really owing to hæmorrhage from a glioma. An unusual seat of the hæmorrhage should make us wary in concluding for ordinary hæmorrhage. Gowers mentions a case of which he says that the soft glioma he found in one case might easily have been taken for a patch of softening.

So now we say that tumour, or any other adventitious product, does not produce optic neuritis in its particular character as this or that kind of pathological product, but in its general character as "a foreign body"; and then, not because it destroys, but by some indirect action.

As it is important to emphasise the distinction betwixt the development of neuritis from a foreign body, and loss of sight from a destructive lesion, certain cases, simple with regard to this matter, may now be mentioned.

In cases of hemiopia there is no atrophy of the discs nor any other change in the fundus, with rare exceptions. The exceptions I have seen were apparent only. In most cases

of hemiopia, in physicians' practice, the lesion is presumably central, for there is nearly always hemiplegia on the side of the blind half-fields. One patient had hemiopia and then neuritis—the neuritis, by the way, adding on, for a time nothing to the defect of sight. But there was tumour of one optic tract, and also a cerebral tumour. In another case there was hemiopia and neuritis; again there was no defect of sight, except as hemiopia implies. This patient's sight, except for hemiopia, was good until his death (Mr. Couper examined this patient's eyes with me). There was no necropsy. The patient had hemiplegia and partial aphasia, and as the hemiplegia came on very gradually, as he had intense pain in the head, he had unquestionably acerebral tumour.

Such a symptom as hemiopia *does* depend on a destructive lesion—often, in physicians' practice, one of some part of the brain. This is quite a different state of things from defect of sight ensuing on changes induced in the optic nerves or their centres by some "foreign body" in the brain, strikingly different when we note that sight may not be affected during such changes. It is all the more strikingly different when, as in the cases of hemiopia instanced, the neuritis may for a time add nothing on to a defect of sight induced by a destructive lesion. It is, however, convenient to glance at this other side of the question. I know of no evidence (post-mortem evidence, I mean) to prove that mere destructive change of any part of the encephalon (excluding, of course, the corpora quadrigemina, optic tracts, and optic nerves) produces permanent loss or even defect of sight. Having regard to Ferrier's recent researches, * I do not deny that defect of sight, or temporary loss of sight, may be caused by destruction of certain parts; but, if a defect of sight does occur in this way, we are not here concerned with it, but with optic neuritis. It may be that a foreign body in Ferrier's visual centre may produce a more intense neuritis, although as yet I know of no evidence in support of the possibility. With the neuritis from some indirect action of a foreign body so seated there may be more defect of sight *from* destruction of the part in question; but this is for the present hypothetical. In the cases of tumour of the middle cerebellar lobe pressing on the corpora quadrigemina, that I have seen, there has been double optic neuritis.

The several different lines of evidence agree in converging to the conclusion that optic neuritis, in cases of intracranial adventitious products, is to be looked on as resulting secondarily from such products in their general character—that of foreign bodies.

* See *Brain*, part xii., "Cerebral Amblyopia and Hemiopia."

Section V.—Various Hypotheses as to the Mode of Production of Changes in the Optic Discs by Intracranial Adventitious Products.

Now comes the question, What is the secondary process? There is the hypothesis of Graefe that one variety of optic neuritis, or of swelling of the disc (*Stauungs-Papilla*) is produced by raised intracranial pressure—the "choked disc" of Clifford Allbutt. It is thus stated by Pagenstecher, who does not adopt it:—"1. Through the increased pressure within the cranium, pressure is brought to bear upon the cavernous sinus, which induces venous congestion in the central vessels of the optic nerve, which is also increased, as regards this, by the scleral ring (von Graefe)." Against this hypothesis is the fact that the tumour or other adventitious product may be very small. A small cortical tumour which cannot raise intracranial pressure to any but the most trifling degree causes double optic neuritis. So may a small tumour of one lateral lobe of the cerebellum or a small one of the middle lobe when there has been but little effusion into the cerebral ventricles. It may, however, be said that neuritis ("descending") most often occurs with small tumours, and that when there is a very large adventitious product, the appearances are those of the "choked disc." But I think it has not yet been shown how we are to distinguish by the ophthalmoscope. According to the hypothesis now under criticism, big tumours should sometimes at least produce a mixture of the two kinds of disc changes; indeed, transitional conditions are spoken of by authorities. Microscopical evidence will perhaps best clear up the dispute.

A small tumour in the cortex has produced just the same kind, and I believe degree, of optic neuritis as a mass of tubercle weighing eleven ounces in one cerebral hemisphere did. In two cases of cerebral tumour already mentioned there was neuritis of the opposite side only. Since any pressure there was would be equal in all directions from a tumour so placed, it is strange that but one eye should suffer from it. Swelling of the discs is not found in cases of vast cerebral hæmorrhage when the intracranial pressure is suddenly greatly increased. Then it may occur in an extreme form when there is no local morbid product of any sort in the brain—that is to say, in cases where there is nothing to raise intracranial pressure. Thus, in one case where, beyond extreme congestion, attributable to mode of death, only microscopical changes in the brain were found, there had been ophthalmoscopically, as

Mr. Couper ascertained for me, "very great swelling and extreme tortuosity of the veins. The distance between the apex and the base of the disc is represented by a + ⅛ glass." I show a drawing by Dr. Gowers, who kindly examined the eyes microscopically for me, of a section of one of the discs. I have seen optic neuritis in a case of cerebral atrophy. Moreover, in some cases the swelling subsides, the discs become atrophic although intracranial pressure is increasing, as is seen in cases of large adventitious products in one cerebral hemisphere or in cases of hydrocephalus along with tumour of the middle lobe of the cerebellum, when the head goes on enlarging. It might seem, at first glance, that in such cases, since the cranial walls yield, there will be compensation. That there is not full compensation is proved in some at least of the cases by the deepening hebetude. Further, there are large cerebral tumours, which destroy life and yet produce no swelling of the discs. Here I would ask if optic neuritis often occurs in cases of large intracranial cancer secondary to cancer elsewhere. One would suppose that in some of these cases the growth would be rapid and the intracranial pressure high. In one case, carefully observed by Dr. Stephen Mackenzie, we found no swelling of the discs and no enlargement of the veins, although post-mortem there was discovered, besides cancer in the thorax, extensive cancer of each cerebral hemisphere and of each lobe of the cerebellum. Then the anatomical objections of free anastomosis by the superior orbital and facial veins, urged by Sesemann, tell strongly against the pressure doctrine.

There is the hypothesis of Schmidt, which ascribes some cases of swollen disc to distension of the optic nerve sheaths. This hypothesis is thus stated by Pagenstecher:— "2. The intracranial pressure must force the fluid from the arachnoid space along the sheath of the optic nerve into the canal-like system present in the lamina cribrosa; and thus swelling, congestion, and the inflammatory symptoms depending upon these, take their origin (Schmidt)." I omit consideration of this part of the subject; the evidence in support of it is at least conflicting. I ask ophthalmic surgeons, What results have been obtained from operations suggested by this hypothesis?

There is the hypothesis of reflex vaso-motor action, generally attributed to Benedict, but, as Gowers points out, first hinted at by Schneller. Objections to this are obvious. In the first place, it might be said that such an hypothesis would explain anything. More particularly we may object to it by asking, Why should the action be upon the vessels

of the optic nerves or their centres in particular? This question is the more important when we note that the disease may be in any part of the encephalon. Yet, on the other hand, we may rejoin, Why does the local gross disease in any part pick out the optic nervous system in any way? Some things here require particular attention.

It is not supposed, of course, by anybody, that the *tumour* provokes contraction, or rather frequent contractions, of the arteries of the optic nerves or centres, but that the contractions result from changes induced by the tumour in grey matter.

It is quite certain that a cortical tumour often does induce changes of instability in grey matter near it, as epileptiform seizures in exact correspondence with its locality demonstrate. The instability induced is one thing, the "foreign bodies" and other pathological changes inducing it are many. It is a warrantable inference that the changes of instability, although induced by the tumour, become independent of it—become autonomous.

Now, it is clear from cases of epileptiform seizures with cortical lesions that the discharges consequent on induced instability produce distant effects—convulsion directly and paralysis indirectly, and continuous paralysis if the fits be repeated. I suggest that there is something analogous in the production of optic neuritis. The analogy, however, is distant. Spasm of big muscles of the skeleton with subsequent paralysis of them is a different thing from there being frequent discharges almost exclusively directed on muscular coats of vessels, and on those only of a couple of nerve bundles, or their centres, or both, occasionally constricting the arteries strongly and leaving them temporarily paralysed. Yet, as already remarked, there occur occasional total failures of sight in patients who have neuritis with good or with only slightly defective sight, and sometimes they occur before neuritis appears. And what is far more to the point, such failures—not always total, however—not unfrequently occur at the onset of epileptiform seizures, that is, when discharge is going on; occurs, we may add, in the cases where optic neuritis is so often found.

I think that optic neuritis may be a doubly indirect result of local gross organic disease; that first there are changes of instability about the tumour; that next these lead on discharges, by intermediation of vaso-motor nerves, to repeated contractions, with subsequent paralyses, of vessels of the optic nerves or centres, and thus, at length, to that trouble of nutrition which is optic neuritis. I repeat that such induced changes are causes of spasm of big muscles. More-

over, in cases of epilepsy proper we see often enough effects produced by discharges on arteries and on viscera, as well as convulsion by discharges on big muscles.

In the attempt to find the method of causation of double optic neuritis by tumour, we ought to consider all the symptoms called non-localising, but which in this connexion are better called symptoms of local gross organic disease. Any acceptable hypothesis must account not for the condition of the disc only, but for the other symptoms also which tumour produces along with that condition. We must bear in mind that optic neuritis and the other symptoms, headache, vomiting, etc., or one or more of them, are often temporary; they may last weeks or months, and may end with the patient's death. But often the headache goes off, the vomiting ceases, the discs clear up either to a quasi-healthy state with good vision or to atrophy. If the illness is acute, the patient may rapidly emaciate during it, and get fat after it. There is an illness, more or less acute. I suppose all the symptoms alluded to are signs, not directly of tumour, but of an encephalitis provoked by the tumour in its character as a foreign body.

That some secondary changes in the brain of some kind are produced by tumour is certain; epileptiform seizures with cortical tumour demonstrate this. As said a moment ago, optic neuritis (and perhaps also the other symptoms) is supposed to be a *doubly indirect* result of tumour. The sequence is supposed to be tumour, changes of instability, effects produced by the latter (hypothetically) on the muscular walled arteries of the optic nerves and centres (certainly in some cases, where the tumour is cortical, on muscles, ordinarily so-called).

Looking from the inside, so to speak, we ought, in our attempts to settle the question as to the process of causation of optic neuritis, to consider all other symptoms which tumour produces, not forgetting, above all, epileptiform seizures. And in such an inquiry it is important to bear in mind that optic neuritis is a pathological condition, and thus that it does not compare in any way, to give an example, with an epileptiform seizure, even if it compares with the internal changes on which that symptom depends.

The hypothesis which seems to me most plausible, when the whole of the non-localising symptoms, and such conditions of the centres as epileptiform seizures imply, are taken into account, is that optic neuritis results doubly indirectly from intracranial tumour by vaso-motor action.

Pagenstecher favours this hypothesis. He writes of it—"However, in the majority of cases we shall do best to con-

tent ourselves with this, if we must have an explanation at all, although it is partially inaccurate, and also requires further confirmation. In my view, then, the affair stands thus: The irritation conveyed through the nerve-tract of the sympathetic to the disc, induces the changes of the nerve-fibres, the hyperæmia, and even the development of new vessels, and in this manner a swelling and obfuscation (*Trübung*) of the disc and the adjacent parts of the retina is brought about. The latter may then for its part have, as a consequence, an extreme degree of congestion of the venous system of the retina.

"In any case it is very easy to bring the analytical facts in accordance with this explanation; and the very often asserted facts of the swelling of the optic disc of one eye only are most easily explicable on this hypothesis."

As to swelling of but one eye, I think, for my part, that it is a difficulty in the way of this hypothesis.

Of course we ought to take into account what has been determined by microscopical examination, especially in cases of swelling of the discs in tubercular meningitis, the facts of which cases we cannot fit into the vaso-motor hypothesis without great hardihood. I do not think that if it were to be proved that there is a "descending change" in neuritis from tumour, the hypothesis of vaso-motor action is thereby invalidated. At any rate, I think it is not invalidated in cases where the foreign body supposed to somehow cause the neuritis is far away from the "optic nervous system," unless, of course, changes can be traced from the part in which the tumour lies—say, for an example, from the lateral lobe of the cerebellum in the case of abscess therein seated. Any way, the opinions of those who have done the hard work of microscopical examination are to be received with great respect. I omit speaking of this important part of my subject, partly because I have no work of my own of the kind to bring forward, and partly because Gowers, Garlick, Nettleship, Brailey, and others will be able from the microscopical point of view to speak with authority for or against any or the whole of the hypotheses stated. Regardless of all hypotheses as to mode of production of optic neuritis, minute pathological research is evidently of great value in enabling us to find out what it means in different cases of intracranial disease.

Section VI. - Treatment

In one regard it may seem absurd to speak of treatment of optic neuritis. We treat the patient and the internal local disease which we suppose to have caused it. If that local internal disease be syphilitic, we may hope, supposing the changes to be recent, to clear them away by mercurial inunction and iodide of potassium. For my own part I believe these hopes frequently end in disappointment; the so-called cure is often temporary, only a removal of symptoms. Supposing, however, the intracranial disease to be a non-syphilitic tumour. According to current opinions we can do nothing for that. But many of the so-called symptoms of tumour, including optic neuritis, are symptoms indirectly produced by tumour. Although two of the hypotheses mentioned as to the causation of optic neuritis are utterly different, all three agree in this, that the changes in the optic nerves are secondary. Although produced by central disease, the changes when produced become independent of that disease—become autonomous. The analogy is not a good one, but I may instance in illustration that when ear disease has produced cerebral abscess, that abscess is then independent of the ear disease; so much so that, if we were able to restore the ear to an entirely normal condition, we should by that do nothing at all for cure of the abscess. Similarly, if we were able to reverse all central changes which had led to optic neuritis, the optic neuritis would, I submit, remain for treatment. Optic neuritis is an independent thing for treatment, although there may remain also another thing for treatment, in encephalitis induced by the central disease. That the neuritis does become autonomous seems clear, by, in some cases, the ensuing of atrophy, which is permanent, when all other signs of central disease are gone, when the central disease has become quiescent. There are people remaining blind from the effects of cerebral tumour who have ceased to show any other signs of any cerebral disease. The central disease has produced changes at a distance and "has left them there." Further, to treat optic neuritis is not to treat a symptom; very often, indeed, there is that pathological condition without any attendant defect of sight. Furthermore we see cases in which optic neuritis is our only clue into their nature—often all we know there is, whatever we may infer.

It is obvious that the best time for treatment is when the neuritis is in its earliest stage, in the præ-amaurotic stage, although it is certain that recovery may follow treatment of

neuritis when sight is almost lost. In the præ-amaurotic stage the acute process, whatever it is, and however determined, has not damaged many nerve-fibres. Hence the importance of the routine examination of the eyes whenever a patient has any nervous symptom, especially headache. Sight fails, I believe, nearly always in a later stage—in a stage less hopeful, but yet not hopeless, for treatment. There would be fewer blind people if the ophthalmoscope were used in some so-called bilious attacks, in some so-called cases of hysteria, etc. The ophthalmic surgeon nearly always sees neuritis in a later stage than a physician usually sees it—in a less hopeful stage. I have many times seen it, when treated early, pass off; and many times have I been consulted for a later stage, with blindness, for which I have usually done little good. I always give iodide of potassium, whether I suspect syphilis or not, and frequently prescribe mercurial inunction in addition.

I know of no treatment which does any good in atrophy, partial or total, after neuritis; it seems difficult to suppose that drugs can do anything. On this matter I ask what ophthalmic surgeons in their larger experience have found, and in particular if strychnia has been of any use in such cases of atrophy.

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